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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Apr 08  "Ask CAS" for self-help around the clock
 NEWS  3  Apr 09  BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS  4  Apr 09  ZDB will be removed from STN
 NEWS  5  Apr 19  US Patent Applications available in IFICDB, IFIPAT, and
IFIUDB
 NEWS  6  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and
ZCAPLUS
 NEWS  7  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS  8  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS  9  Jun 03  New e-mail delivery for search results now available
 NEWS 10  Jun 10  MEDLINE Reload
 NEWS 11  Jun 10  PCTFULL has been reloaded
 NEWS 12  Jul 02  FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22  USAN to be reloaded July 28, 2002;
                  saved answer sets no longer valid
 NEWS 14  Jul 29  Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30  NETFIRST to be removed from STN
 NEWS 16  Aug 08  CANCERLIT reload
 NEWS 17  Aug 08  PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
 NEWS 19  Aug 19  Aquatic Toxicity Information Retrieval (AQUIRE)
                  now available on STN
 NEWS 20  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 21  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 22  Aug 26  Sequence searching in REGISTRY enhanced
 NEWS 23  Sep 03  JAPIO has been reloaded and enhanced
 NEWS 24  Sep 16  Experimental properties added to the REGISTRY file
 NEWS 25  Sep 16  Indexing added to some pre-1967 records in CA/CAPLUS
 NEWS 26  Sep 16  CA Section Thesaurus available in CAPLUS and CA
 NEWS 27  Oct 01  CASREACT Enriched with Reactions from 1907 to 1985

 NEWS EXPRESS  October 14 CURRENT WINDOWS VERSION IS V6.01,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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FILE 'HOME' ENTERED AT 11:38:34 ON 17 OCT 2002

=> file medline caplus biosis embase scisearch agricola

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FILE 'AGRICOLA' ENTERED AT 11:38:59 ON 17 OCT 2002

=> s gastric acid secretion
L1 26873 GASTRIC ACID SECRETION

=> s l1 (p) inhibit?
L2 13071 L1 (P) INHIBIT?

=> s l2 (a) fat
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L8 (A) FAT'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L9 (A) FAT'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L10 (A) FAT'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L11 (A) FAT'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L12 (A) FAT'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L13 (A) FAT'
L3 371 L2 (A) FAT

=> s l2 (p) fat
L4 343 L2 (P) FAT

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=> d uplicate remove l4
'UPLICATE' IS NOT A VALID FORMAT
'REMOVE' IS NOT A VALID FORMAT
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in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
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REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> duplicate remove l4
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L4
L5          132 DUPLICATE REMOVE L4 (211 DUPLICATES REMOVED)

=> s l5 and py<2000
   3 FILES SEARCHED...
   5 FILES SEARCHED...
L6          124 L5 AND PY<2000

=> s l6 (p) (milk or egg)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L31 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L33 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L35 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L37 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L39 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L41 (P) '
L7            1 L6 (P) (MILK OR EGG)

=> d l7 1 ibib abs

L7   ANSWER 1 OF 1     MEDLINE
ACCESSION NUMBER:    91304125     MEDLINE
DOCUMENT NUMBER:     91304125   PubMed ID: 2072799
TITLE:               Diet and nutrition in ulcer disease.
AUTHOR:              Marotta R B; Floch M H
CORPORATE SOURCE:    Nutrition Support Service, Norwalk Hospital, Connecticut.
SOURCE:              MEDICAL CLINICS OF NORTH AMERICA, (1991 Jul) 75
                     (4) 967-79.  Ref: 58
                     Journal code: 2985236R. ISSN: 0025-7125.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
                     General Review; (REVIEW)
                     (REVIEW, ACADEMIC)
LANGUAGE:            English
FILE SEGMENT:        Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:         199108
ENTRY DATE:          Entered STN: 19910908
                     Last Updated on STN: 19910908
                     Entered Medline: 19910822
AB   In this era of H2-inhibitors, the available evidence does not
     support the need to place peptic ulcer disease patients on restrictive
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diets. The major goal of diet is to avoid extreme elevations of **gastric acid secretion** and the direct irritation of gastric mucosa. In view of this, only slight modifications in the patient's usual diet are recommended. Table 1 depicts a sample menu for chronic peptic ulcer disease. Frequent **milk** ingestion as previously prescribed is not encouraged. This is owing to the transient buffering effect and significant **gastric acid secretion** effect of **milk**. The **fat** content of **milk** has no influence on these effects. Spices, in particular black pepper, red pepper, and chili powder, may produce dyspepsia. One study shows red chili powder to have no detrimental effect on duodenal ulcer healing. It has also been proposed that daily pepper ingestion may have a beneficial adaptive cytoprotective response. While still controversial and under evaluation, peptic ulcer patients should avoid any spice that causes discomfort, especially during exacerbation of peptic disease. Currently, studies indicate that it is prudent to avoid alcohol. This is especially true for the concentrated forms, such as 40% (80 proof) alcohol. Coffee should be avoided on the basis of its strong acid secretagogue property. Coffee can induce dyspepsia. Whether noncoffee caffeine-containing beverages (tea, soft drinks) induce peptic ulcer is unknown, but they are acid secretion stimulators. Decaffeinated coffee has an acid stimulating effect as well. It is reasonable to have peptic ulcer patients restrict decaffeinated coffee and all caffeine-containing beverages. There appears to be no evidence to restrict dietary fiber. Some fiber-containing foods may possess factors that are protective against ulcer disease. According to the Mayo Clinic Diet Manual, previously recommended small frequent feedings have not been shown to be more effective than three meals per day in the treatment of chronic peptic ulcer disease. This reference cites authorities advising against extra feedings because of increased acid secretion and unnecessary complication of eating patterns. However, some patients claim to be relieved of symptoms with more frequent feedings, especially during acute phases. Citric acid juices may induce reflux and cause discomfort in selective patients. Stomach distention with large quantities of food should be discouraged. Although there is now little role for dietary therapy, one should note that bland and ulcer diets probably are not detrimental to most persons if they are used for a short time and may have some psychological benefit.(ABSTRACT TRUNCATED AT 400 WORDS)

=> d his

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 11:38:59 ON 17 OCT 2002

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L1         26873 S GASTRIC ACID SECRETION
L2         13071 S L1 (P) INHIBIT?
L3           371 S L2 (A) FAT
L4           343 S L2 (P) FAT
L5           132 DUPLICATE REMOVE L4 (211 DUPLICATES REMOVED)
L6           124 S L5 AND PY<2000
L7             1 S L6 (P) (MILK OR EGG)
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=> s l1 (p) inhibitor
L8 3514 L1 (P) INHIBITOR

=> s l8 (p) fat
L9 64 L8 (P) FAT

=> duplicate remove l9
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
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PROCESSING COMPLETED FOR L9
L10 24 DUPLICATE REMOVE L9 (40 DUPLICATES REMOVED)

=> d l10 1-24 ibib abs

L10 ANSWER 1 OF 24 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:618851 CAPLUS
DOCUMENT NUMBER: 137:179683
TITLE: Ranitidine and omeprazole as adjuvant therapy to pancrelipase to improve fat absorption in patients with cystic fibrosis
AUTHOR(S): Francisco, Mary Pat; Wagner, Mary H.; Sherman, James M.; Theriaque, Douglas; Bowser, Ellen; Novak, Donald A.
CORPORATE SOURCE: Department of Pediatrics, University of Florida, Gainesville, FL, 32610, USA
SOURCE: Journal of Pediatric Gastroenterology and Nutrition (2002), 35(1), 79-83
CODEN: JPGND6; ISSN: 0277-2116
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Inadequate treatment of pancreatic insufficiency in patients with cystic fibrosis (CF) causes malabsorption of nutrients with significant sequelae.
The objective of this study was to measure the effect of acid suppressant therapy on fat absorption in patients with CF who received a pH-sensitive, enteric-coated microtablet enzyme product. A double-blind, placebo-controlled crossover study of 12 children and 10 adults with pancreatic insufficient CF was performed. All subjects were receiving pancrelipase therapy (Pancrease MT10 and MT16; Ortho-McNeil, Springhouse, PA, U.S.A.) and for the study also received either placebo or ranitidine (Zantac; Glaxo-Wellcome, Research Triangle Park, NC U.S.A.) 5 mg/kg or 10 mg/kg daily. The adult subjects also received omeprazole therapy (Prilosec; AstraZeneca/Merck, Wilmington, DE, U.S.A.), 20 mg daily, as adjuvant therapy to pancreatic enzymes. Serial 3-day fat-balance studies were performed in the Clin. Research Center. The data were analyzed using individual paired t tests that compared each treatment with placebo and two repeated-measures, general linear model F tests. The linear model for all subjects showed no overall adjuvant drug effect on fat absorption, P = 0.32. A second linear model F test anal. of adult subjects, comparing all four drug treatments (placebo, ranitidine 5 and 10 mg/kg daily and omeprazole), also showed no difference in fat absorption, P = 0.15.

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Paired t test subgroup anal. of the adults showed an improvement of 4.97% (P = 0.003) in mean fat absorption comparing low-dose ranitidine to placebo. All other t test analyses showed no significant change in fat absorption between placebo and acid suppressant treatment. There was marked intersubject and intrasubject variability in fat absorption. No overall significant improvement in fat absorption could be demonstrated with adjuvant therapy. Fat absorption measured by 3-day fat-balance studies varied greatly even when comparing the same subject for placebo and baseline treatments, despite identical dietary fat and enzyme intakes.

The large variability limited our ability to test for a difference in fat absorption and has significant implication for the use of this test, considered the gold std., for detg. enzyme dosage adequacy.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 24 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:873248 CAPLUS
DOCUMENT NUMBER: 136:20253
TITLE: Preparation of peptides as drugs such as antihypertensives, analgesics, gastric acid-secretion inhibitors, and growth hormone production inhibitors
INVENTOR(S): Sakamoto, Kenji
PATENT ASSIGNEE(S): Nagoshi, Hideo, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 17 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2001335596	A2	20011204	JP 2000-152459	20000524

AB (Poly)peptides having activity to reduce blood pressure, analgesic activity, activity to inhibit prodn. of growth hormone, activity to inhibit accumulation of **fat** in **fat** cells, activity to increase calcium level in blood, activity to inhibit secretion of gastric acid, activity to increase prodn. of prostaglandin E2 by prostaglandin E2-producing cells, activity to stimulate proliferation of osteoblasts, and activity to increase prodn. of growth hormone by growth hormone-producing cells are prepd. They provides antihypertensives, analgesics, growth hormone prodn. **inhibitors**, **fat** accumulation **inhibitors**, increasers for blood calcium concn., **gastric acid-secretion inhibitors**, promoters for prostaglandin E prodn., promoters for proliferation of osteoblastic cell, and promoters for growth hormone prodn., which have mechanisms of actions different from prior art drugs. These (poly)peptides are also useful for the treatment of osteoporosis and dwarfism. Thus, H-Gln-Arg-Gly-Thr-Gln-Lys-Ser-Ile-Ile-Ile-His-Thr-Ser-Glu-Asp-Gly-Lys-Val-OH, which was prepd. by a peptide synthesizer, exhibited analgesic activity in a hot plate assay on rats.

L10 ANSWER 3 OF 24 MEDLINE DUPLICATE 1

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ACCESSION NUMBER: 2001336175 MEDLINE
DOCUMENT NUMBER: 21296949 PubMed ID: 11403540
TITLE: Gastric acid suppression and treatment of severe exocrine pancreatic insufficiency.
AUTHOR: DiMagno E P
CORPORATE SOURCE: Department of Internal Medicine, Mayo Clinic, 200 First Street S.W., Rochester, MN 55905, USA.
SOURCE: Best Pract Res Clin Gastroenterol, (2001 Jun) 15 (3) 477-86. Ref: 20
Journal code: 101120605. ISSN: 1521-6918.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010917
Entered Medline: 20010816

AB Adding either H(2)-receptor antagonists (cimetidine or ranitidine) or proton pump **inhibitors** to an adequate amount of lipolytic activity improves **fat** malabsorption in most cases and abolishes steatorrhoea in up to 40% of children and adults with cystic fibrosis and in adults with chronic pancreatitis. Acid suppression improves **fat** absorption because the resultant increase in pH within the upper gastrointestinal tract improves the survival of lipolytic activity, reduces duodenal volume flow and prevents the precipitation of bile acids.

These effects increase the concentration of intraduodenal lipolytic activity and promote the aggregation of bile acids and the micellar solubilization of lipid. The amount of lipase that should be recommended is controversial, but we interpret our studies as indicating that at least 90 000 United States Pharmacopeia (USP) units should be ingested with meals. This amount of lipolytic activity taken with an agent that suppresses **gastric acid secretion** improves **fat** absorption in most patients and may even abolish steatorrhoea.

L10 ANSWER 4 OF 24 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000270041 MEDLINE
DOCUMENT NUMBER: 20270041 PubMed ID: 10807887
TITLE: Role of lipase in the regulation of postprandial **gastric acid secretion** and emptying of **fat** in humans: a study with orlistat, a highly specific lipase **inhibitor**.
AUTHOR: Borovicka J; Schwizer W; Guttmann G; Hartmann D; Kosinski M; Wastiel C; Bischof-Delaloye A; Fried M
CORPORATE SOURCE: Gastroenterology and Nuclear Medicine Departments, University Hospitals, Lausanne and Zurich, Switzerland.
SOURCE: GUT, (2000 Jun) 46 (6) 774-81.
Journal code: 2985108R. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

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ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000811
Last Updated on STN: 20000811
Entered Medline: 20000801

AB BACKGROUND AND AIMS: To investigate the importance of lipase on gastric functions, we studied the effects of orlistat, a potent and specific **inhibitor** of lipase, on postprandial gastric acidity and gastric emptying of **fat**. METHODS: Fourteen healthy volunteers participated in a double blind, placebo controlled, randomised study. In a two way cross over study with two test periods of five days, separated by at least 14 days, orlistat 120 mg three times daily or placebo was given with standardised daily meals. In previous experiments we found that this dose almost completely inhibited postprandial duodenal lipase activity. Subjects underwent 28 hour intragastric pH-metry on day 4, and a gastric emptying study with a mixed meal (800 kcal) labelled with (999m)Tc sulphur colloid (solids) and (111In)thiocyanate (**fat**) on day 5. Gastric pH data were analysed for three postprandial hours and the interdigestive periods. RESULTS: Orlistat inhibited almost completely (by 75%) lipase activity and accelerated gastric emptying of both the solid (by 52%) and **fat** (by 44%) phases of the mixed meal ($p<0.03$). Orlistat increased postprandial gastric acidity (from a median pH of 3.3 to 2.7; $p<0.01$). Postprandial cholecystokinin release was lower with orlistat ($p<0.03$). CONCLUSION: Lipase has an important role in the regulation of postprandial **gastric acid secretion** and **fat** emptying in humans. These effects might be explained by lipolysis induced release of cholecystokinin.

L10 ANSWER 5 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:485557 BIOSIS
DOCUMENT NUMBER: PREV200000485557
TITLE: The impact of omeprazole on children with cystic fibrosis (CF) who require high-dose pancreatic enzymes: A pilot study.
AUTHOR(S): Chung, Y. (1); Gunasekaran, T. S. (1); Angst, D. B. (1); Blue, B. (1); VandenBranden, S. (1)
CORPORATE SOURCE: (1) Department of Pediatrics, Divisions of Pediatric Pulmonology and Gastroenterology, Lutheran General Children's Hospital, Park Ridge, IL USA
SOURCE: JPGN, (2000) Vol. 31, No. Supplement 2, pp. S73. print. Meeting Info.: World Congress of Pediatric Gastroenterology, Hepatology, and Nutrition Boston, Massachusetts, USA August 05-09, 2000
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 6 OF 24 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1998208456 MEDLINE
DOCUMENT NUMBER: 98208456 PubMed ID: 9548675
TITLE: Pancreatic dysfunction and treatment options.
AUTHOR: Nakamura T; Takeuchi T; Tando Y
CORPORATE SOURCE: Third Department of Internal Medicine, Hirosaki University School of Medicine, Aomori, Japan.
SOURCE: PANCREAS, (1998 Apr) 16 (3) 329-36. Ref: 95
Journal code: 8608542. ISSN: 0885-3177.

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PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980609
Last Updated on STN: 19980609
Entered Medline: 19980527

AB Pancreatic steatorrhea and pancreatic diabetes are the dominant symptoms of patients in the decompensated stage of chronic pancreatitis (CP). In this stage, the nutritional state is greatly disturbed and hypoglycemia and labile infection are involved. Pancreatic enzyme replacement therapy is the principal treatment method for pancreatic steatorrhea. Before initiating this therapy, dietary **fat** intake must be determined and pancreatic lipase and bicarbonate secretion function must be evaluated. Upper small intestinal pH is regulated by **gastric acid secretion**, and abnormal gastric emptying changes lipolysis. In addition, precipitation of bile acids in the upper small intestine and ileal brakes due to undigested **fats** and carbohydrates must be considered. Porcine pancreatin, bacterial lipase, and acid-resistant fungal lipase are used as enzymes for replacement therapy. Conventional, entero-coating, and enteric-coated microsphere preparations of porcine pancreatin are available for treatment and are formulated to protect against gastric acids, to dissolve enzymes at optimum pH, and to be emptied simultaneously with food from the stomach. **Gastric acid secretion** suppressants, such as H2 blockers or a proton pump **inhibitor**, can also be used concomitantly with pancreatin preparations. In consideration of both strengths and weaknesses of these preparations, types and dosages of enzyme replacement therapy should be carefully prescribed, and fecal **fats** should be examined repeatedly by a simple and rapid method during treatment. Attention should also be paid to changes in body weight and nutritional indices (e.g., nutritional parameters, **fat**-soluble vitamins). The relationship between carbohydrate maldigestion/malabsorption in CP patients and treatment of pancreatic diabetes are topics for future research.

L10 ANSWER 7 OF 24 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96407470 MEDLINE
DOCUMENT NUMBER: 96407470 PubMed ID: 8811523
TITLE: Twenty-four hour ambulatory gastric and duodenal pH profiles in cystic fibrosis: effect of duodenal hyperacidity on pancreatic enzyme function and fat absorption.
AUTHOR: Barraclough M; Taylor C J
CORPORATE SOURCE: Department of Paediatrics, University of Sheffield, England.
SOURCE: JOURNAL OF PEDIATRIC GASTROENTEROLOGY AND NUTRITION, (1996 Jul) 23 (1) 45-50.
Journal code: 8211545. ISSN: 0277-2116.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128

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Last Updated on STN: 19970128
Entered Medline: 19961216

AB Overt steatorrhoea remains a problem for some patients with cystic fibrosis (CF) despite supraphysiological dosages of pancreatic enzymes. As pancreatin release and enzyme function is influenced by duodenal pH, we have used 24-h ambulatory pH measurements to assess the extent and duration of postprandial hyperacidity. Readings were obtained from the stomach and proximal duodenum in 16 CF patients (aged 6 months to 12 years) using a dual-channel antimony electrode. The fasting gastric and duodenal pH values were normal in all patients (mean pH values of 1.3, and 6.8, respectively). There was, however, a marked drop in duodenal pH in the first postprandial hour, which became more pronounced with successive meals. The total time that duodenal pH was < 5 varied from 15 to 90% of the recording (mean 57%). Overnight the duodenal pH returned to normal levels. A subgroup of five patients were studied before and after treatment with omeprazole, a potent **inhibitor** of **gastric acid secretion**. There were significant improvements in both weight gain and **fat** absorption. This study supports the hypothesis that the postprandial duodenal pH is excessively acid in patients with CF and may be an important element in the continuing **fat** malabsorption experienced by some patients. This malabsorption may limit the efficacy of the newer high-lipase pancreatic enzyme supplements and lead to delayed enzyme release, a possible factor in the recent reports of proximal colonic strictures.

L10 ANSWER 8 OF 24 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95047170 MEDLINE
DOCUMENT NUMBER: 95047170 PubMed ID: 7958703
TITLE: Intracisternal injection of apolipoprotein A-IV inhibits gastric secretion in pylorus-ligated conscious rats.
AUTHOR: Okumura T; Fukagawa K; Tso P; Taylor I L; Pappas T N
CORPORATE SOURCE: Department of Surgery, Duke University Medical Center, Durham, North Carolina.
CONTRACT NUMBER: DK 32288 (NIDDK)
DK 38216 (NIDDK)
DK 44072 (NIDDK)
+
SOURCE: GASTROENTEROLOGY, (1994 Dec) 107 (6) 1861-4.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941227

AB BACKGROUND/AIMS: **Fat** feeding increases not only serum but also cerebrospinal fluid concentration of apolipoprotein (apo) A-IV, a protein produced mainly by the small intestine in the rat. We hypothesized that apo A-IV may have a central effect on gastric secretion. METHODS: Gastric juice was collected by the pylorus ligation method. Rats underwent pylorus ligation and received intracisternal injection of apo A-IV under brief isoflurane anesthesia. Two hours after the injection, gastric juice was collected and gastric acid output determined. RESULTS: Intracisternal

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injection of 0.5 microgram apo A-IV had no effect on gastric secretion. However, **gastric acid secretion** was significantly inhibited by intracisternal injection of 1 microgram apo A-IV. Furthermore, intracisternal administration of higher doses of apo A-IV (2.0 and 4.0 microgram) resulted in greater inhibition of **gastric acid secretion** in a dose-dependent manner. On the contrary, 4 micrograms of apo A-I intracisternally injected failed to inhibit **gastric acid secretion**. Intraperitoneal administration of 15 micrograms of apo A-IV did not alter gastric secretion. CONCLUSIONS: These results suggest that apo A-IV may act in the brain to inhibit **gastric acid secretion**. Apo A-IV might be a central enterogastrone, which is a gastric **inhibitor** produced by the small intestine in response to **fat** feeding.

L10 ANSWER 9 OF 24 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 94241510 MEDLINE
DOCUMENT NUMBER: 94241510 PubMed ID: 8185155
TITLE: Integration of postprandial function in the proximal gastrointestinal tract. Role of CCK and sensory pathways.
AUTHOR: Raybould H E; Lloyd K C
CORPORATE SOURCE: CURE/UCLA Digestive Diseases Research Center, VA West Los Angeles.
CONTRACT NUMBER: DDK 41004 (NIDDK)
DK 41301 (NIDDK)
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1994 Mar 23) 713 143-56. Ref: 37
Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 19940621
Last Updated on STN: 19990129
Entered Medline: 19940615

AB Cholecystokinin (CCK) stimulates vagal afferent fiber discharge, both gastric and intestinal, which seems to result in reflex decrease in gastric motility, **gastric acid secretion**, and stimulation of pancreatic protein secretion. Endogenous release of CCK by **fat** or soybean trypsin **inhibitor** also alters function by way of a capsaicin-sensitive pathway. We suggest that CCK is released locally from the intestine and acts locally or systemically to stimulate vagal afferent fiber discharge to alter proximal gastrointestinal function (Fig. 14). In this way, in addition to its effect on food intake, CCK and the neural pathway integrate function in the proximal gastrointestinal tract, regulating the entry of food into the duodenum to ensure effective digestion and absorption.

L10 ANSWER 10 OF 24 MEDLINE
ACCESSION NUMBER: 91304125 MEDLINE
DOCUMENT NUMBER: 91304125 PubMed ID: 2072799
TITLE: Diet and nutrition in ulcer disease.

HBM

AUTHOR: Marotta R B; Floch M H
CORPORATE SOURCE: Nutrition Support Service, Norwalk Hospital, Connecticut.
SOURCE: MEDICAL CLINICS OF NORTH AMERICA, (1991 Jul) 75 (4) 967-79.
Ref: 58
Journal code: 2985236R. ISSN: 0025-7125.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199108
ENTRY DATE: Entered STN: 19910908
Last Updated on STN: 19910908
Entered Medline: 19910822

AB In this era of H2-**inhibitors**, the available evidence does not support the need to place peptic ulcer disease patients on restrictive diets. The major goal of diet is to avoid extreme elevations of **gastric acid secretion** and the direct irritation of gastric mucosa. In view of this, only slight modifications in the patient's usual diet are recommended. Table 1 depicts a sample menu for chronic peptic ulcer disease. Frequent milk ingestion as previously prescribed is not encouraged. This is owing to the transient buffering effect and significant **gastric acid secretion** effect of milk. The **fat** content of milk has no influence on these effects. Spices, in particular black pepper, red pepper, and chili powder, may produce dyspepsia. One study shows red chili powder to have no detrimental effect on duodenal ulcer healing. It has also been proposed that daily pepper ingestion may have a beneficial adaptive cytoprotective response. While still controversial and under evaluation, peptic ulcer patients should avoid any spice that causes discomfort, especially during exacerbation of peptic disease. Currently, studies indicate that it is prudent to avoid alcohol. This is especially true for the concentrated forms, such as 40% (80 proof) alcohol. Coffee should be avoided on the basis of its strong acid secretagogue property. Coffee can induce dyspepsia. Whether noncoffee caffeine-containing beverages (tea, soft drinks) induce peptic ulcer is unknown, but they are acid secretion stimulators. Decaffeinated coffee has an acid stimulating effect as well. It is reasonable to have peptic ulcer patients restrict decaffeinated coffee and all caffeine-containing beverages. There appears to be no evidence to restrict dietary fiber. Some fiber-containing foods may possess factors that are protective against ulcer disease. According to the Mayo Clinic Diet Manual, previously recommended small frequent feedings have not been shown to be more effective than three meals per day in the treatment of chronic peptic ulcer disease. This reference cites authorities advising against extra feedings because of increased acid secretion and unnecessary complication of eating patterns. However, some patients claim to be relieved of symptoms with more frequent feedings, especially during acute phases. Citric acid juices may induce reflux and cause discomfort in selective patients. Stomach distention with large quantities of food should be discouraged. Although there is now little role for dietary therapy, one should note that bland and ulcer diets probably are not detrimental to most persons if they are used for a short time and may have some psychological benefit.(ABSTRACT TRUNCATED AT 400

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WORDS)

L10 ANSWER 11 OF 24 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 91300965 MEDLINE
DOCUMENT NUMBER: 91300965 PubMed ID: 2070700
TITLE: Intracolonic fat inhibits gastric acid secretion independent of gastrin release in the dog.
AUTHOR: Hashimoto T; Lluis F; Gomez G; Hill F L; Greeley G H Jr; Thompson J C
CORPORATE SOURCE: Department of Surgery, University of Texas Medical Branch, Galveston 77550.
CONTRACT NUMBER: 5R37 DK 15241 (NIDDK)
P01 DK 35608 (NIDDK)
SOURCE: DIGESTIVE DISEASES AND SCIENCES, (1991 Jul) 36 (7) 888-92. Journal code: 7902782. ISSN: 0163-2116.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199108
ENTRY DATE: Entered STN: 19910908
Last Updated on STN: 19980206
Entered Medline: 19910821

AB The purpose of this study was to examine the effect of perfusion of the colon with a fatty acid (oleic acid) on peptone-stimulated **gastric acid secretion** and release of gastrin in conscious dogs. **Gastric acid secretion** was monitored by continuous intragastric titration. Perfusion of the colon with sodium oleate (24 mmol/hr) inhibited **gastric acid secretion** (14.2 +/- 2.6 meq/hr) stimulated by a peptone meal (1%) significantly (P less than 0.05) when compared to perfusion of the colon with saline alone (20.1 +/- 1.6 meq/hr). The serum elevation in gastrin in response to intragastric instillation of the peptone meal was not affected by the colonic perfusion of oleic acid. Plasma concentrations of peptide YY (PYY) increased significantly in response to perfusion of the colon with saline or sodium oleate, and the integrated release of PYY in response to sodium oleate [6.9 +/- 2.8 ng (60-120) min/ml] was significantly greater than the response to saline [3.1 +/- 0.7 ng (60-120) min/ml]. The results of this study indicate that inhibition of **gastric acid secretion** by perfusion of the colon with **fat** is not due to an inhibition of gastrin release. In addition, because PYY is an **inhibitor** of **gastric acid secretion**, it is possible that PYY participates as an **inhibitor** of **gastric acid secretion** by the colon.

L10 ANSWER 12 OF 24 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 90338917 MEDLINE
DOCUMENT NUMBER: 90338917 PubMed ID: 2380653
TITLE: Glucagon-like peptide-1 (7-36)-NH2: a physiological inhibitor of gastric acid secretion in man.
AUTHOR: O'Halloran D J; Nikou G C; Kreymann B; Ghatei M A; Bloom S R
CORPORATE SOURCE: Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London.

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SOURCE: JOURNAL OF ENDOCRINOLOGY, (1990 Jul) 126 (1) 169-73.
Journal code: 0375363. ISSN: 0022-0795.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 19901012
Last Updated on STN: 19901012
Entered Medline: 19900907

AB Glucagon-like peptide (GLP)-1 (7-36)-NH2 is a peptide found in the mucosal endocrine cells of the intestine, and plasma levels of GLP-1 (7-36)-NH2 immunoreactivity show a rise after the ingestion of a **fat** or mixed-component meal. We investigated the effects of physiological infusion of GLP-1 (7-36)-NH2 on a submaximal **gastric acid secretion** in healthy volunteers at a rate known to mimic the observed postprandial rise in plasma concentrations. Corrected gastric acid output decreased to less than 50% and volume output to 33% of stimulated values. After the infusion, the secretion of gastric acid recovered immediately to preinhibition values. These results suggest a novel role for GLP-1 (7-36)-NH2 as a physiological **inhibitor** of **gastric acid secretion** in man.

L10 ANSWER 13 OF 24 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 90255820 MEDLINE
DOCUMENT NUMBER: 90255820 PubMed ID: 2340960
TITLE: Cholecystokinin in the inhibition of gastric secretion and gastric emptying in humans.
AUTHOR: Konturek S J; Kwiecien N; Obtulowicz W; Kopp B; Oleksy J; Rovati L
CORPORATE SOURCE: Institute of Physiology, Academy of Medicine, Krakow, Poland.
SOURCE: DIGESTION, (1990) 45 (1) 1-8.
Journal code: 0150472. ISSN: 0012-2823.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199006
ENTRY DATE: Entered STN: 19900720
Last Updated on STN: 19900720
Entered Medline: 19900627

AB Cholecystokinin (CCK) is known to inhibit **gastric acid secretion** and gastric emptying but its physiological role in the inhibition of gastric functions is not settled. In this study performed on 16 young male subjects, **gastric acid secretion** and emptying rate were determined after intragastric administration of 8% peptone meal alone or in combination with intravenous infusion of graded doses of CCK-8 (5-80 pmol/kg.h) or with addition of vegetable oil to meal without or with pretreatment with loxiglumide, a specific CCK antagonist. CCK-8 infusion at lower dose (5 pmol/kg.h) was ineffective but at higher doses (20-80 pmol/kg.h) it resulted in a significant reduction in acid output by 39 and 43% and a decrease in gastric emptying from 54% to 40 and 22%, respectively. Pretreatment with loxiglumide abolished almost

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completely the inhibition of both gastric acid and gastric emptying by CCK-8. **Fat** added to peptone meal reduced **gastric acid secretion** by 42-65% and decreased gastric emptying to 24-32%. The pretreatment with loxiglumide tended to reduce **fat**-induced inhibition of **gastric acid secretion** and gastric emptying but the difference in the inhibition of gastric functions between the tests without and with loxiglumide was not significant. This study provides evidence that exogenous CCK administered at pharmacological doses is a potent **inhibitor** of **gastric acid secretion** and gastric emptying and probably acts via specific CCK receptors. In contrast, **fat** induces inhibition of **gastric acid secretion** and gastric emptying that cannot be fully attributed to hormonally acting CCK.

L10 ANSWER 14 OF 24 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 89240637 MEDLINE
DOCUMENT NUMBER: 89240637 PubMed ID: 2654927
TITLE: Pancreatic juice enhances fat-stimulated release of enteric hormones in dogs.
AUTHOR: Lluis F; Gomez G; Hashimoto T; Fujimura M; Greeley G H Jr; Thompson J C
CORPORATE SOURCE: Department of Surgery, Hospital Santa Creu i Sant Pau, Universidad Autonoma de Barcelona, Spain.
CONTRACT NUMBER: 5R37 DK 15241 (NIDDK)
PO1 DK 35608 (NIDDK)
SOURCE: PANCREAS, (1989) 4 (1) 23-30.
Journal code: 8608542. ISSN: 0885-3177.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198906
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19980206
Entered Medline: 19890620

AB The presence of pancreatic juice in the intestinal lumen results in the hydrolysis of dietary **fat**. The hydrolytic products of dietary **fat** are potent stimulants of pancreatic exocrine secretion and potent **inhibitors** of **gastric acid secretion**. In this study, residual pancreatic enzyme activity in the intestinal lumen may account for the observed increase of triglyceride-stimulated pancreatic exocrine secretion and the release of peptides during diversion of pancreatic juice. The presence of pancreatic juice enhanced the pancreatic protein output that was stimulated by the intraduodenal administration of a triglyceride (corn oil, 2 g/kg/h) by 240% (p less than .05). The presence of pancreatic juice during the intraduodenal administration of a triglyceride nearly abolished the output of gastric acid as well as the release of gastrin (p less than .05) that had been stimulated by the intragastric placement of a 10% peptone meal. Pancreatic juice in the duodenum significantly enhanced the triglyceride-stimulated release of cholecystokinin-33/39, secretin, neurotensin, peptide YY, pancreatic polypeptide, and insulin (p less than .05) when compared with the release of these enteropancreatic hormones during the diversion of pancreatic juice. This study shows that the presence of pancreatic juice in the duodenal lumen enhances the

fat-stimulated release of enteric hormones that have a stimulatory action on the enteroacinar and enteroinsular axis as well as an inhibitory action (enterogastrone-like activity) on the postprandial regulation of gastric function.

L10 ANSWER 15 OF 24 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 88084275 MEDLINE
DOCUMENT NUMBER: 88084275 PubMed ID: 2891586
TITLE: Somatostatin may not be a hormonal messenger of fat-induced inhibition of gastric functions.
AUTHOR: Mogard M H; Maxwell V; Wong H; Reedy T J; Sytnik B; Walsh J H
CORPORATE SOURCE: Center for Ulcer Research and Education, Veterans Administration Wadsworth Medical Center, Los Angeles, California.
CONTRACT NUMBER: DK 17294 (NIDDK)
DK 17328 (NIDDK)
DK 35445 (NIDDK)
SOURCE: GASTROENTEROLOGY, (1988 Feb) 94 (2) 405-8.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198802
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19880220

AB The present study was designed to evaluate somatostatin as a hormonal **inhibitor** of gastric functions in humans. Seven healthy volunteers were investigated on 6 separate days. Peptone meal-stimulated **gastric acid secretion** was measured by intragastric titration for 2 h and gastric emptying was estimated with a dye-dilution technique. The effect of intravenous administration of somatostatin at 0, 12.5, 50, 100, and 200 pmol/kg.h was investigated and related to the effect of intragastric administration of 100 ml of vegetable oil. Plasma somatostatinlike immunoreactivity was elevated during intravenous administration of somatostatin at 100 and 200 pmol/kg.h, whereas no increase was detected in response to the oil. Somatostatin infusion at 100 and 200 pmol/kg.h significantly inhibited the acid secretion by 25% and 65%, and the oil reduced the acid output by 41%. Somatostatin at 100 and 200 pmol/kg.h significantly enhanced gastric emptying, whereas the oil inhibited gastric emptying. These observations suggest that somatostatin may not be an important hormonal messenger of **fat**-induced inhibition of acid secretion or gastric emptying.

L10 ANSWER 16 OF 24 MEDLINE
ACCESSION NUMBER: 87018632 MEDLINE
DOCUMENT NUMBER: 87018632 PubMed ID: 2876506
TITLE: Physiological role of somatostatin in the digestive tract: gastric acid secretion, intestinal absorption, and motility.
AUTHOR: Krejs G J

HBM

SOURCE: SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY. SUPPLEMENT, (1986) 119 47-53. Ref: 32
Journal code: 0437034. ISSN: 0085-5928.
PUB. COUNTRY: Norway
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198611
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19950206
Entered Medline: 19861117

AB Somatostatin is found in both endocrine cells and nerve fibres of the gastrointestinal tract and has several inhibitory effects on the digestive tract. Somatostatin is a potent **inhibitor** of gastrin release; its secretion is regulated predominantly by the cholinergic pathway, which inhibits somatostatin and thus stimulates gastrin release. **Gastric acid secretion** is inhibited by both the paracrine and circulating peptide (hormonal) effects of somatostatin. Somatostatin secretion is a direct effect of acid on the somatostatin cell, since it is unaffected by the axonal blocker tetrodotoxin. Somatostatin antiserum eliminates the inhibitory effect of somatostatin and thus augments acid secretion. It therefore appears that somatostatin plays a physiological role in regulating **gastric acid secretion**, and it is possible that a lack of the inhibitory function of somatostatin is an aetiological factor in peptic ulcer disease. Postprandially, a rise in serum somatostatin concentration occurs which is twice as high with protein and **fat** as it is with carbohydrates. Several studies have shown that somatostatin inhibits nutrient absorption, indicating that somatostatin might be a physiological regulator in the homeostasis of ingested nutrients by modulating the intestinal absorption rate. Experiments have also demonstrated that somatostatin infusion inhibits intestinal motility; the interval between migrating myoelectric complexes is increased, and transit time is increased.

L10 ANSWER 17 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 86181904 EMBASE
DOCUMENT NUMBER: 1986181904
TITLE: Physiological role of somatostatin in the digestive tract: Gastric acid secretion, intestinal absorption, and motility.
AUTHOR: Krejs G.J.
CORPORATE SOURCE: University of Texas Health Science Center at Dallas, Southwestern Medical School, Dallas, TX, United States
SOURCE: Scandinavian Journal of Gastroenterology, Supplement, (1986) 21/SUPPL. 119 (47-53).
CODEN: SJGSB8
COUNTRY: Norway
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
048 Gastroenterology
029 Clinical Biochemistry
003 Endocrinology
LANGUAGE: English

HBM

AB Somatostatin is found in both endocrine cells and nerve fibres of the gastrointestinal tract and has several inhibitory effects on the digestive tract. Somatostatin is a potent **inhibitor** of gastrin release; its secretion is regulation predominantly by the cholinergic pathway, which inhibits somatostatin and thus stimulates gastrin release. **Gastric acid secretion** is inhibited by both the paracrine and circulating peptide (hormonal) effects of somatostatin. Somatostatin secretion is a direct effect of acid on the somatostatin cell, since it is unaffected by the axonal blocker tetrodotoxin. Somatostatin antiserum eliminates the inhibitory effect of somatostatin and thus augments acid secretion. It therefore appears that somatostatin plays a physiological role in regulating **gastric acid secretion**, and it is possible that a lack of the inhibitory function of somatostatin is an aetiological factor in peptic ulcer disease. Postprandially, a rise in serum somatostatin concentration occurs which is twice as high with protein and **fat** as it is with carbohydrates. Several studies have shown that somatostatin inhibits nutrient absorption, indicating that somatostatin might be a physiological regulator in the homeostasis of ingested nutrients by modulating the intestinal absorption rate. Experiments have also demonstrated that somatostatin infusion inhibits intestinal motility; the interval between migrating myoelectric complexes is increased, and transit time is increased.

L10 ANSWER 18 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1983:294184 BIOSIS
DOCUMENT NUMBER: BA76:51676
TITLE: PREDICTION OF CIMETIDINE DISPOSITION IN THE AGED.
AUTHOR(S): RITSCHEL W A
CORPORATE SOURCE: UNIV. CINCINNATI MED. CENT., MAIL LOCATION NO. 4, CINCINNATI, OH 45267, USA.
SOURCE: METHODS FIND EXP CLIN PHARMACOL, (1983) 5 (4), 255-262. CODEN: MFEPDX. ISSN: 0379-0355.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The change in the elimination half-life of cimetidine [a potent **inhibitor** of **gastric acid secretion**] as a function of age can accurately be predicted by an equation previously published. For the age-dependent change of the apparent volume of distribution, a correction factor was developed based on total body fluid and body **fat** as a function of age. The predicted values correlate well with experimental data reported. A computer program was developed for cimetidine dosage regimen prediction which can be used for normal subjects, young and geriatric patients with and without renal impairment.

L10 ANSWER 19 OF 24 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:155604 CAPLUS
DOCUMENT NUMBER: 98:155604
TITLE: On the neurohumoral interrelations in the regulation of gastric secretion
AUTHOR(S): Shlygin, G. K.
CORPORATE SOURCE: Inst. Nutr., Moscow, USSR
SOURCE: Agressologie (1982), 23(6), 245-8

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CODEN: AGSOA6; ISSN: 0002-1148
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Gastric acid secretion** by dogs was stimulated by i.v. infusions of mixts. of L-amino acids and by most individual amino acids, with the exception of dicarboxylic acids that inhibited the secretory function of the stomach. The stimulatory effect of amino acids was blocked by atropine, and the inhibitory effect of glutamic acid by adrenoblockers, indicating that the amino acids act via neural mechanisms. Although atropine inhibited acid secretion 5-6 h following a protein meal, it was not affected by gastrin **inhibitors**. Gastrin is known to play a major role in acid secretion induced by **fat** ingestion. Thus, **gastric acid secretion** after eating is influenced both by the response to absorbed amino acids and by gastrin, and the predominant mechanism is dependent on the chem. nature of nutrients in food.

L10 ANSWER 20 OF 24 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 81024926 MEDLINE
DOCUMENT NUMBER: 81024926 PubMed ID: 7419011
TITLE: Effect of intravenous lipid on gastric acid secretion stimulated by intravenous amino acids.
AUTHOR: Varner A A; Isenberg J I; Elashoff J D; Lamers C B; Maxwell V; Shulkes A A
CONTRACT NUMBER: AM 17328 (NIADDK)
SOURCE: GASTROENTEROLOGY, (1980 Nov) 79 (5 Pt 1) 873-6.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198012
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19980206
Entered Medline: 19801218
AB Intraduodenal **fat** is a potent **inhibitor** of all forms of **gastric acid secretion** in humans. Studies were performed in random order on 3 separate days in 5 normal subjects to determine if intravenous **fat** (Intralipid) altered **gastric acid secretion** stimulated by intravenous amino acids in humans. Mean (+/- SE) gastric acid output during a 4-hr intravenous amino acid infusion (21 g L-amino acids; Freamine II) plus glucose (50 g. to maintain isocaloric and isoosmolar solutions) was 43.2 +/- 3.2 meq/4 hr. Intraduodenal **fat** fusion (20 g of Intralipid) significantly ($P < 0.02$) suppressed amino acid-stimulated acid output. Interestingly, intravenous **fat** (20 g of Intralipid) also significantly ($P < 0.02$) inhibited acid secretion (14.8 +/- 6.3 meq/4 hr); similar to the effect observed with intraduodenal **fat** (12.7 +/- 4.9 meq/4 hr). Serum levels of CCK, gastrin, and GIP were measured at 30-min intervals throughout each study. Cholecystokinin and GIP increased significantly from basal during intraduodenal **fat** infusion. There were no other changes in serum CCK, gastrin, or GIP during any of the other tests. It is concluded that in normal subjects intravenous **fat** is a potent **inhibitor** of

intraveous amino acid-stimulated **gastric acid secretion**, similar in effect to intraduodenal **fat**. The inhibitory effect of intravenous **fat** on amino acid-stimulated **gastric acid secretion** is probably not mediated by release of either CCK of GIP. Circulating **fat** may play a role in the control of some forms of **gastric acid secretion**.

L10 ANSWER 21 OF 24 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 79025442 MEDLINE
DOCUMENT NUMBER: 79025442 PubMed ID: 100366
TITLE: Effect of duodenal fat on plasma levels of gastrin and secretin and on gastric acid responses to gastric and intestinal meals in dogs.
AUTHOR: Rayford P L; Konturek S J; Thompson J C
SOURCE: GASTROENTEROLOGY, (1978 Nov) 75 (5) 773-7.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19781227

AB The effects of duodenal instillation of sodium oleate (10 mmoles per hr) on plasma levels of gastrin and secretin and on **gastric acid secretion** in response to gastric and intestinal meals were determined. Four dogs prepared with a septum between stomach and duodenum were provided with a special cannula that allowed separate access to the stomach or duodenum. Each dog received a 10% liver extract meal introduced either into the stomach (gastric phase) or into the duodenum (intestinal phase). Sodium oleate administered during the gastric phase caused approximately a 30% reduction in plasma gastrin level and a 25% inhibition of **gastric acid secretion**. Sodium oleate given during the intestinal phase completely abolished the plasma gastrin response and resulted in a 75% inhibition of **gastric acid secretion**. Plasma secretin levels were not changed during the gastric phase or the intestinal phase by instillation of sodium oleate. These results show that **fat** in the duodenum is a potent **inhibitor** of gastrin release and **gastric acid secretion**; the intestinal mechanism involved does not appear to affect plasma secretin concentrations.

L10 ANSWER 22 OF 24 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 76177949 MEDLINE
DOCUMENT NUMBER: 76177949 PubMed ID: 1265443
TITLE: Jejunal inhibition of pentagastrin-induced gastric acid secretion in man and Heidenhain pouch dogs.
AUTHOR: Christiansen J; Holst J J; Rokkjaer M
SOURCE: SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (1976) 11 (2) 219-24.
Journal code: 0060105. ISSN: 0036-5521.
PUB. COUNTRY: Norway
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

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FILE SEGMENT: Priority Journals
ENTRY MONTH: 197607
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19760706

AB The administration of hypertonic glucose and saline and of **fat** intrajejunally in man caused a marked and almost identical inhibition of pentagastrin-stimulated **gastric acid secretion** . Hypertonic glucose administered intrajejunally in Heidenhain pouch dogs resulted in an equal inhibition of pentagastrin-induced acid secretion from the pouch and the main stomach, whereas hypertonic saline had no effect. The study demonstrates the existence of potent jejunal **inhibitors** of gastric secretion, which seem to operate independently of vagal innervation.

L10 ANSWER 23 OF 24 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 15
ACCESSION NUMBER: 1975:54836 CAPLUS
DOCUMENT NUMBER: 82:54836
TITLE: Release of gastric inhibitor from the intestine of dogs infused intravenously with sodium oleate
AUTHOR(S): Kowalewski, K.; Secord, D. C.
CORPORATE SOURCE: Surg. Med. Res. Inst., Univ. Alberta, Edmonton, Alberta, Can.
SOURCE: Pharmacology (1974), 12(3), 177-85
CODEN: PHMGBN
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Lyophilized exts. of canine intestinal secretion or mucosa were prepd. using dogs infused i.v. for 6 hr with saline or Na oleate. The exts. were assayed for their gastric acid inhibitory action by i.v. infusion into Heidenhain pouches of dogs stimulated with pentagastrin. **Gastric acid secretion** was inhibited only in dogs infused with Na oleate. Thus, the i.v. infusion of **fat** results in the release of a gastric **inhibitor** of intestinal origin that behaves as an enterogastrone.

L10 ANSWER 24 OF 24 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1970:1938 CAPLUS
DOCUMENT NUMBER: 72:1938
TITLE: Blockade of norepinephrine uptake and other activities of 5-(3'-dimethylaminopropyl)dibenzo[a,d][1,4]cyclohep tadiene hydrochloride (AY-8794) and structurally related compounds
AUTHOR(S): Lippmann, Wilbur
CORPORATE SOURCE: Biogenic Amines Lab., Ayerstt Lab., Montreal, Que., Can.
SOURCE: Biochem. Pharmacol. (1969), 18(10), 2517-29
CODEN: BCPCA6
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of AY-8794 [5-(3-dimethylaminopropyl)-dibenzo[a,d][1,4]cycl oheptadiene-HCl] and structurally related compds. on the uptake of norepinephrine into the storage sites and other activities were detd. AY-8794 blocks the uptake of 3H labeled norepinephrine into the mouse and rat heart. AY-8794 is a potent **inhibitor** of **gastric**

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acid secretion in the rat. The free fatty acid mobilization in vitro from minced rat epididymal **fat** pads induced by norepinephrine is inhibited at a high level and is stimulated at a low level of AY-8794. AY-8794 exhibits antiinflammatory activity in the rat. The structural requirements for various of these activities were detd.

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(FILE 'HOME' ENTERED AT 11:38:34 ON 17 OCT 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 11:38:59 ON 17 OCT 2002

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L1         26873 S GASTRIC ACID SECRETION
L2         13071 S L1 (P) INHIBIT?
L3           371 S L2 (A) FAT
L4           343 S L2 (P) FAT
L5           132 DUPLICATE REMOVE L4 (211 DUPLICATES REMOVED)
L6           124 S L5 AND PY<2000
L7             1 S L6 (P) (MILK OR EGG)
L8          3514 S L1 (P) INHIBITOR
L9            64 S L8 (P) FAT
L10           24 DUPLICATE REMOVE L9 (40 DUPLICATES REMOVED)
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FULL ESTIMATED COST	48.42	48.63

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